pericardial drainage may keep the patient alive while he is being transferred to the operating theater; this fact is especially relevant when cardiothoracic surgery facilities are not available in the hospital to which the patient has been referred.

CASE REPORT

A 48-year-old male patient with bronchial asthma antecedents was hospitalized with retrosternal pain. Results of physical examination, ECG, and thoracic radiography were normal. At 36 hours after hospital admission, he presented with intense thoracic and abdominal pain followed by hypotension and cardiac arrest.

Cardiopulmonary resuscitation was applied and an electromechanical dissociation was demonstrated. After pericardiocentesis and aspiration of 20 ml of blood, pulse recovery was achieved.

Echocardiogram showed dilation of the ascending aorta with intimal flap, as well as a hemopericardium with CT symptoms.

Subsequently, upon a new episode of electromechanical dissociation, a pericardial catheter was inserted. Blood was aspirated from the pericardium, resulting in pulse and systolic artery pressure recovery to 80 to 100 mm Hg.

As no cardiothoracic surgery facilities were available in the hospital, the patient was transferred to a University Hospital at 100-km distance for the respective treatment. During the transfer, the patient was monitored and further blood aspirations of 5 to 20 ml through the pericardial catheter were performed, when systolic arterial pressure was below 80 mm Hg, with consequential hemodynamic improvement.

At the University Hospital, angiography evidenced a DeBakey type I AD with rupture into the pericardial cavity. The patient was treated surgically, but he died on the third postoperative day due to associated complications.

DISCUSSION

Aortic dissection associated with rupture into the pericardial cavity may have a lethal outcome, if the patient does not receive immediate surgical treatment. In some cases, placing a pericardial drainage catheter can keep the patient functional while he is being transferred to the operating theater. It is, however, essential to avoid any brusque decompression of the pericardial cavity, as well as an excessive increase of arterial pressure, because there exists the risk of increasing aortic tear due to the enhanced stress on the aortic walls, which could cause the patient's death.

In cases of massive pericardial bleeding, autotransfusion, as described by Aravot and Vidne, may prove to be the method of choice, aspirating blood from the pericardial sac and retransfusing it through a central venous catheter.

In conclusion, for patients with AD with pericardial tamponade, pericardial drainage may have life-saving effects during transportation to the operating theater, which may be of vital importance when the patient has been referred to a hospital lacking a cardiac surgery unit and he has to be transferred to another hospital.

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Acetaminophen-induced Eosinophilic Pneumonia*

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We present the first case (to our knowledge) of acetaminophen-induced eosinophilic pneumonia. Although the patient had taken an acetaminophen-containing combination drug, Kinuya-chinnetsu, for only three days, the abnormal shadow on his chest radiograph continued for about a month. Lymphocyte stimulation tests showed that both Kinuya-chinnetsu and acetaminophen induced the proliferation of peripheral blood lymphocytes. (Chest 1993; 104:291-92)

CRP = C-reactive protein; \( n_v = \) normal value

Eosinophilic pneumonia is caused by many kinds of drugs, such as chlorpropamide, sulfonamides, penicillin, and nitrofurantoin. In this report, we describe a patient with eosinophilic pneumonia, peripheral blood eosinophilia, and skin rash after administration of an acetaminophen-containing combination drug. To our knowledge, this is the first report of acetaminophen-induced eosinophilic pneumonia.

CASE REPORT

A 63-year-old man was admitted to our hospital because of fever, dyspnea, and skin rash. Two weeks prior to hospital admission, he had flu-like symptoms and had taken a combination drug for the common cold, Kinuya-chinnetsu, containing acetaminophen (37.5 percent), bromvalerylurea (25 percent), caffeine (12.5 percent), lactose (12.5 percent), and potato starch (12.5 percent), for three days. Five days later, he had development of skin rash and dyspnea. He had no known allergies in his history.

Physical examination at the time of hospital admission revealed coarse crackles in both lungs and marked skin rash. His body temperature was 38.8°C.

Laboratory data revealed peripheral eosinophilia of 13 percent (normal value [\( n_v \]): 0 to 2 percent) with leukocytosis (white blood cells: 14,800/mm\(^3\) [\( n_v \): 3,700 to 8,800/mm\(^3\)]), erythrocyte sedimentation rate (ESR) of 110 mm/h (\( n_v \): <10 mm/h), C-reactive protein (CRP) of 12.9 mg/dl (\( n_v \): <0.25 mg/dl), and hypoxemia (PaO\(_2\): 54.6 mm Hg, and PaCO\(_2\): 33.4 mm Hg). The concentration of IgE was 3,490 IU/ml (\( n_v \): <570 IU/ml). Chest radiograph at the time of

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CHEST / 104 / 1 / JULY, 1993 291
hospital admission revealed bilateral infiltrates and hilar lymphadenopathy (Fig 1, A). Spirometry revealed a combination impairment (percent VC: 63 percent and FEV, %: 54 percent). The recovery of bronchoalveolar lavage was 57 percent (86 ml/150 ml), where the cell count was $1.88 \times 10^5$ cells per milliliter (nv: 0.8 to 1.2 $\times 10^5$ cells per milliliter), with 26.1 percent macrophages (nv: >90 percent), 44.4 percent lymphocytes (nv: <10 percent), 27.1 percent eosinophils (nv: <1.0 percent), and 2.4 percent neutrophils (nv: <2.0 percent). The peripheral lung tissue sections obtained by transbronchial lung biopsy showed alveolar septal fibrosis and predominant eosinophilic infiltration, indicating eosinophilic pneumonia.

Without any drugs, his rash resolved on the second hospital day. Though his dyspnea and fever also resolved on the 17th day, his chest radiograph revealed worsened interstitial shadows and loculated effusion in the minor fissure (Fig 1, B). After administration of 30 mg/d of prednisolone for a week and 30 mg every other day for another week, his chest radiograph showed clearing of the infiltrates, and the eosinophilia disappeared. All the levels of ESR, CRP, blood gases, and IgE became normal.

Lymphocyte stimulation test performed seven days after the discontinuation of prednisolone therapy, according to the method of Paronetto and Popper, was positive for Kinuya-chinnetsu and acetaminophen (Table 1). From these results, his illness was diagnosed as acetaminophen-induced eosinophilic pneumonia.

**Table 1—Lymphocyte Stimulation Test**

<table>
<thead>
<tr>
<th>Drug</th>
<th>cpm</th>
<th>SI*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>505</td>
<td>100</td>
</tr>
<tr>
<td>Kinuya-chinnetsu</td>
<td>2,179</td>
<td>432</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>1,126</td>
<td>223</td>
</tr>
<tr>
<td>Bromvalerylurea</td>
<td>768</td>
<td>152</td>
</tr>
<tr>
<td>Caffeine</td>
<td>778</td>
<td>154</td>
</tr>
<tr>
<td>Kinuya-chingaijo</td>
<td>510</td>
<td>101</td>
</tr>
<tr>
<td>Nabumetone</td>
<td>646</td>
<td>128</td>
</tr>
</tbody>
</table>

*Stimulation index (percent) (negative <200, positive ≥200).

**DISCUSSION**

Acetaminophen, one of the cyclooxygenase inhibitors, was synthesized in 1878, and it has been broadly used as an antipyretic analgesic since 1948. Acetaminophen is now a well-known anodyne, contained in many kinds of combination drugs for the common cold, and it is known as a highly safe agent when used in therapeutic doses. Overdose causes liver or myocardial damage. There are several reports of acetaminophen-induced asthma in aspirin-sensitive patients. However, we could not find any reports of acetaminophen-induced eosinophilic pneumonia. Although eosinophilic pneumonia has been reported secondary to treatment with various drugs, including many kinds of anodynes, this patient suggests that acetaminophen can cause eosinophilic pneumonia.

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